Experimental Studies on Control of

OAK WILT DISEASE

E. B. Himelick and Howard W. Fox

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EXPERIMENTAL STUDIES ON CONTROL OF OAK WILT DISEASE

By E. B. Himelick and Howard W. Fox[1]

The forest land of our country was once believed to be an almost inexhaustible natural resource. The increase in the human population as well as its migration has brought about a gradual, and in some regions an almost complete, conversion of timbered land to cultivated land. Man has placed tremendous pressure on the remaining forested land by his demand for lumber and other forest products. Also, because the public is becoming more outdoor-minded and is increasing its use of natural forested areas for recreational purposes, more attention must be given to these areas by governmental agencies if they are to be preserved, maintained, and developed for the future.

The necessity for preserving our forested areas has intensified research in the fields of both insect and disease control. Over the last 60 years the chestnut blight disease has killed almost all of the chestnut trees in the eastern and midwestern parts of the United States. Also, more recently, elm phloem necrosis and Dutch elm disease threaten to destroy the entire American elm population. Considerable losses of oak timber and oak shade trees from the oak wilt disease have occurred in the last 10 to 20 years in Wisconsin, Iowa, Minnesota, and Illinois. In some areas in Wisconsin and Iowa, the mortality of the oak timber due to the oak wilt disease is 50 percent or more. In Illinois, where 56 percent of the total forest stand is oak, the disease threatens to completely devastate vast forested areas of the state.

Formation of the North Central Regional Oak Wilt Technical Committee in 1951 stimulated laboratory and field research on the oak wilt disease. The sharing of information among various research agencies and financial support given by the National Oak Wilt Research Committee, and by county, state, and federal agencies, have accounted for the major advances that have been made in the knowledge of this tree disease.

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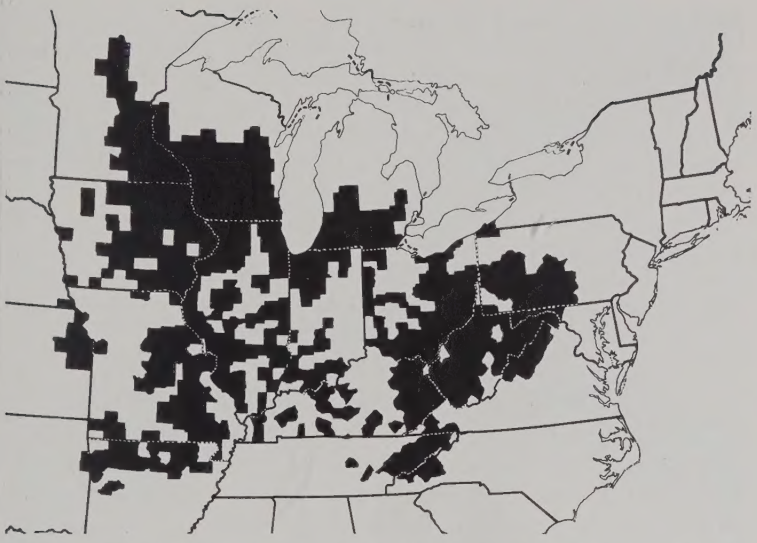
HISTORY OF THE OAK WILT DISEASE

The oak wilt disease is believed to have originated in the upper Mississippi Valley of the United States. One of the earliest accounts of oaks dying, which might have been attributable to the oak wilt disease, was made by Warder (1881) near Madison, Wisconsin. The gradual dying of black oaks, a few at a time over a period of years, was thought at that time to have been caused by drouth. Later, Chapman (1915), working in Minnesota on the life history of the insect *Agrilus bilineatus,* reported several areas of trees of the red oak group which had died from an unknown cause. These deaths, however, were thought to have been caused by Armilaria root rot. The symptoms of the disease, described in Chapman's report, corresponded very closely to symptom expression on trees infected with the oak wilt fungus. Many foresters and farmers in Illinois, Wisconsin, and Iowa recall observing the death of oak trees in the early 1920's in a manner similar to the way oaks die in an active oak wilt area. Oak wilt was given as a common name for the disease in the early observations and experimental work in Wisconsin.

After plant pathologists began working on the disease in 1940, a fungus was isolated and named *Chalara quercina* Henry (1944). Bretz (1952) observed the production of a sexual stage and renamed the fungus *Endoconidiophora fagacearum* Bretz. Mycological work by Hunt (1956) necessitated changing the scientific name to *Ceratocystis fagacearum* (Bretz) Hunt.

The oak wilt disease is believed to be native to the United States and is not known to occur in any other country. In the United States the disease is known to be present in 19 states (Fig. 1). By estimating the age of diseased oaks in the Ozarks and eastern states, substantial evidence has been obtained to indicate that the disease was widely disseminated by the mid-1940's. Movement of the disease from the north central states has been toward the south and east in the United States. In Illinois oak wilt is known to be present in 78 of the 102 counties. It is prevalent in the northern and western counties in the state and occurs in scattered areas throughout the southern and eastern counties. The more concentrated areas of oak wilt occur along the major rivers in the north half of the state and 30 to 40 miles inland from Lake Michigan.

Known hosts of the oak wilt fungus include 36 species of oak and six species closely related to oak. All species of oak tested have been proved susceptible to the fungus either by natural infection or by



Oak wilt distribution by counties in the United States as of 1960. (Fig. 1)

artificial inoculation. The six susceptible species closely related to oak are: Chinese chestnut, *Castanea mollissima* Bl.; American chestnut, *C. dentata* Borkh.; Spanish chestnut, *C. sativa* Mill.; Allegheny chinquapin, *C. Pumila* Mill.; tanbark-oak, *Lithocarpus densiflorus* Rehd.; and bush chinquapin, *Castanopsis sempervirens* Dudl. (Bretz, 1951, 1952a, 1953, 1955, 1957; Ernst and Bretz, 1953). Several investigators are testing native and introduced species of oak for resistance to the disease. As yet no species has proved resistant.

SYMPTOMS

In early stages of the disease in the red oak group, wilting and browning of the foliage usually appear in the upper crown. Wilting progresses downward until the entire tree is affected. Browning of leaves begins at the apex and lobes of the leaf blade and progresses to the midrib. Partial to complete defoliation accompanies foliar symptoms. Frequently brown discoloration may be found in the outer sapwood of wilting branches and in the cambial area of the trunk of infected trees. Infected trees generally die in one growing season. No trees of the red oak group have been observed to have survived more than two years after initial symptoms of oak wilt were noted.

Symptoms expressed by infected trees in the white oak group are varied. Leaves on individual branches turn yellow and remain attached to the branches. Leaves on the outer tips of branches usually wilt the first year. In succeeding years large branches may die back resulting in a "stag-headed" appearance. Some trees succumb to the disease in one to three years. As much as one-third to one-half of the infected trees of the white oak group appear to completely recover.

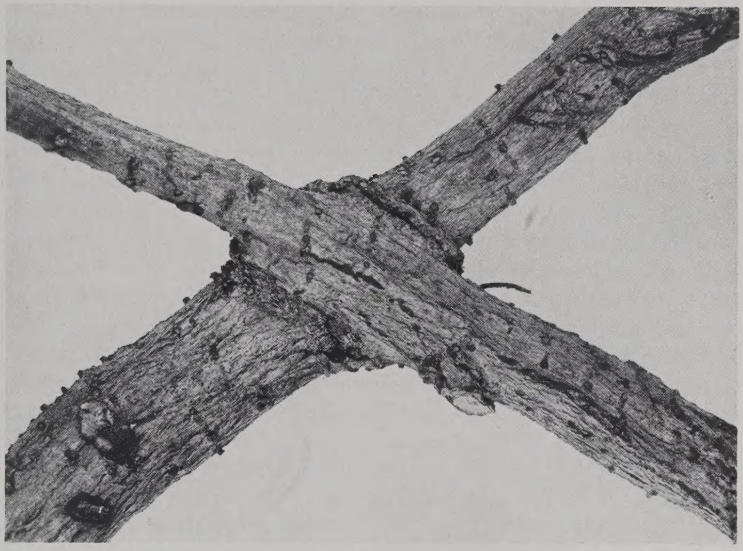
Foliage symptoms of the oak wilt disease on trees of the red oak group are sufficiently distinctive so that an experienced person can usually recognize the disease without culturing for the fungus. However, infected trees of the white oak group usually cannot be recognized without using laboratory procedures.

METHODS OF SPREAD OF THE DISEASE

Macroscopic development of the oak wilt fungus occurs as a fungus mat which is found beneath the loosened bark on some diseased trees. It is believed that the fungus mats are an important source of inoculum in the transmission of the fungus to healthy trees. Asexual spores of the fungus are usually abundant on mature fungus mats produced on diseased trees and may remain viable for as long as 330 days on air-dried mats (Curl, 1955). A characteristic fruit odor, which is usually present, attracts certain sap-feeding insects and squirrels to the fungus mats. Cracks in the bark over the fungus mats allow a portal of entry for the attracted insects. Oak wilt fungus mats have not been reported to occur on naturally infected trees of the white oak group.

Successful experimental transmission of *Ceratocystis fagacearum* to oaks by different species of insects in the family *Nitidulidae* has been reported by several workers (Norris, 1953; Dorsey *et al.*, 1953; Himelick *et al.*, 1954; Thompson *et al.*, 1955; McMullen *et al.*, 1955, 1960; and Jewel, 1956). Rodent feeding on the fruiting mats of *C. fagacearum* in forests of Illinois has been observed, and experimental evidence indicates that fox and gray squirrels are possible vectors of the oak wilt fungus (Himelick and Curl, 1955).

Also, it is possible that beetles which breed in diseased oaks may, upon emergence, carry viable spores of the fungus on or in their bodies. Experimental transmission of the fungus through feeding wounds inflicted by the flat-headed borer, *Chrysobothris femorata,* was reported by Himelick and Curl (1958). Also, Buchanan (1958) obtained successful transmission by the small oak bark beetle, *Pseudopityophthorus minutissimus.*



A typical root graft which was found by excavating between two oak trees in northern Illinois. (Fig. 2)

Pathologists in Wisconsin reported that local spread of the oak wilt fungus between diseased and healthy trees was possible through grafted roots between adjacent trees (Knutz and Riker, 1950). They found abundant natural grafting by digging and washing out the roots of black oaks. A typical root graft found by excavating between two oak trees in northern Illinois is shown in Fig. 2. Investigators have demonstrated experimentally that poisons and dyes readily pass from the root system of one tree to another through these natural unions. Root grafting between trees of the red and white oak groups is believed to be uncommon. White oaks in a mixed stand often remain unaffected, even though the number of red and black oaks in the area have been substantially reduced by the oak wilt disease.

PREVIOUS STUDIES ON CONTROL OF THE DISEASE

A few preliminary reports have been published by workers in the various states on the results of control measures that are being used (Drake, 1956; Hershberger, 1956; Murphy, 1954; True *et al.*, 1955; and Kuntz, 1954). Control of the oak wilt disease seems particularly

feasible in the eastern states where relatively few areas are affected. In the midwestern states, however, where the disease is widely distributed, control on a statewide basis would be very costly unless a method can be used which is efficient, economical, and simple.

General statewide control efforts are being carried out in seven of the 19 states having oak wilt, and another four are studying control measures in localized areas (Hepting, 1955). One of the most complete reports published is a five-year summary of a control program in Missouri by Jones and Bretz (1958). They tested three different methods: isolation of infected trees by poisoning all oaks within 50 feet of infected trees, sanitation by cutting and burning diseased trees, and a combination of both treatments. A recent report of control work in Pennsylvania indicates that the incidence of oak wilt disease has not been materially reduced in areas where treatments had been carried on for seven years (Craighead and Nelson, 1960).

Control measures in experimental use today involve several procedures. The objective of most agencies doing experimental control work is to limit the potential inoculum for overland spread of the fungus by girdling, poisoning, and sometimes felling and burning infected trees. In conjunction with these procedures, some agencies are either poisoning healthy trees to prevent root-graft spread, or they are trenching with large trenching machines around infected trees to sever any roots that may be grafted to healthy trees.

One of the early control measures to prevent mat formation was carried out in Pennsylvania by wholesale peeling of diseased trees (Morris, 1955). Boyce (1954a) reported that mats were produced on at least half of the infected trees left standing, but only on 11 percent of the felled trees. Morris and Fergus (1952) had reported the same success earlier. Later studies by Morris (1955) indicated that felling, deep girdling, and poisoning with sodium arsenite were all good means for preventing mat formation. He reported that fungus mats were seldom produced on trees felled in early stages of wilt, particularly if the trees were felled in early summer. If felling of diseased trees was delayed six weeks after complete defoliation, approximately one-third of them had mats produced on their trunks. Boyce (1954b) obtained a large reduction in the number of living insects inhabiting fungus mats by spraying with an insecticide.

Burning of infected trees is not always practical in forested areas. Not only is burning a hazard during the summer, but it is very costly. A method recommended by Hepting (1955) for treating infected city trees was that of felling, bucking up the large portions, and burning the

small branches. This method requires that the large portions be sprayed with an insecticide to prevent insect attack and the stump should be destroyed by burning.

Spilker and Young (1955) found that *C. fagacearum* was short-lived (three to four weeks) in lumber blocks subjected to temperatures of 25 and 27.5° C. However, at 5° C. the fungus was still viable after 12 weeks. Longevity of the fungus in the wood also was related to the amount of moisture — as the lumber dried the longevity of the fungus decreased. Brandt (1953) reported that oak wilt has no adverse effect on the strength of oak wood. Therefore, logs may be harvested for lumber or for other purposes.

Control of underground spread through root grafts between diseased and healthy oaks was first studied in Wisconsin. Kuntz and Riker (1951) reported two control methods: poisoning of healthy trees surrounding diseased trees, and the use of a tractor-drawn knife to mechanically sever root connections between diseased and healthy trees. Root severing was done by drawing a 3-foot knife through the soil with a large tractor. Yount (1955, 1958) found that the oak wilt fungus can live three years or more in diseased roots of infected trees. He also showed that the length of time between inoculation and the first wilt symptoms was longer for root-inoculated trees than for trunk-inoculated trees.

Various chemicals have been used for poisoning oak trees. The chemical used most frequently is 2,4,5-T (2,4,5-trichlorophenoxyacetic acid). Nichols (1957) reported that 2,4,5-T at 16 pounds active ingredient per 100 gallons in kerosene used as a basal bark treatment usually resulted in 100 percent stem-kill on oak. According to Nichols, the most consistently effective treatments used in timber improvement studies were 2,4,5-T in kerosene and sodium arsenite applied in frills.

Wilcox *et al.* (1956) reported that a solution of 20 to 40 percent sodium arsenite was the most effective chemical tested in their experiments for debarking. Loosening of the bark on infected oak trees is a definite advantage in that it prevents fungus mat formation and lessens the chances of overland spread of the disease. Several different studies which involved the hazards of using sodium arsenite were made by Webb *et al.* (1956). They reported that the greatest danger to wildlife was due to careless handling of the chemical in the woods, and the second most dangerous source was from the freshly treated girdles. The danger period for wildlife obtaining a sufficient quantity of poison from treated girdles was reported by them as being less than 12 hours. The use of sodium arsenite with a dye and a deer repellent and the

careful handling and dispensing of the chemical will eliminate any possibility of danger. Incorporating creosote derivatives, mainly cresylic acid, and a dull, dark dye have proved very effective in reducing the possible attraction to wildlife.

Ammate (ammonium sulfamate) is one of the safest herbicides in use today. However, the killing qualities of Ammate are not comparable to those of 2,4,5-T and sodium arsenite (Nichols, 1957). Drake *et al.* (1957) reported that 2,4-D; 2,4,5-T; mixtures of these; and Ammate gave variable amounts of top-kill. But root-kill was limited, since some sprouting from surviving roots did occur. In the same publication they also reported results using methyl bromide to kill oak roots. Effective root-kill was obtained by injecting the soil fumigant to a depth of 23 to 30 inches within the root zone. No further spread of the disease was observed when the chemical was applied between infected and healthy trees.

LOCATION AND DESCRIPTION OF SINNISSIPPI FOREST, THE AREA OF THE PRESENT STUDY

The present study on oak wilt eradication was made in Sinnissippi Forest in Northern Illinois. The forest, located near Oregon, Illinois, and approximately 40 miles south of the Wisconsin border, has 2,300 acres of timber, of which native hardwood comprises 1,648 acres. Since 1938, management of the forest has been under the supervision of the Department of Forestry of the University of Illinois. Research on the control of oak wilt was started in 1954 in a 360-acre area of the forest. In 1955, control measures were applied to the total area with the exception of 180 acres, which was reserved as a nontreated check and left in its natural state. The 180 acres also served as an area in which the amount of natural spread of the disease could be intensively studied.

Sinnissippi Forest is divided into 40-acre compartments which serve as organizational units to facilitate the operations of intensive management and research activities (Fig. 3). The first forestwide survey and timber cruise of Sinnissippi Forest was completed in 1949 by members of the University of Illinois Forestry Department. A forest type-map was prepared and a timber inventory made. At the time of the survey, age counts were obtained throughout the forest in order to assign stands to 20-year age classes. A second timber cruise was made in the winter of 1953-54.

The species of oak which are found in Sinnissippi Forest are black oak, *Quercus velutina* Lam.; northern red oak, *Q. rubra* L.; northern

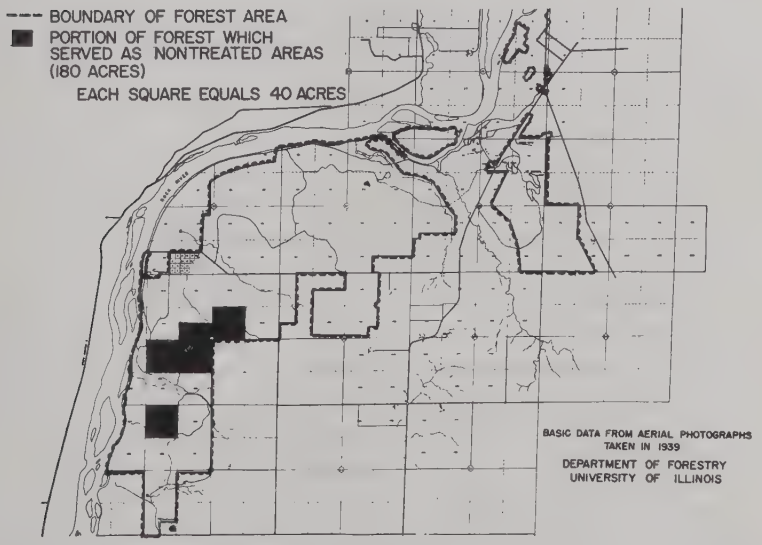
pin oak (Jack or hill's oak), *Q. ellipsoidalis* Hill.; shingle oak (northern laurel oak), *Q. imbricaria* Michx.; white oak, *Q. alba* L.; and bur oak (mossy cup oak), *Q. macrocarpa* Michx.

Since late 1948, improvement cuttings have been made regularly to remove misshapen, overmature, declining, or otherwise undesirable trees. Each year the improvement cut is concentrated on an area equal to one-tenth of the whole forest area. The amount of improvement cut for each area is equivalent to the amount of new growth added in a 10-year period and takes into consideration the loss due to diseases, insects, and other causes. Theoretically, this method of selective cutting enables the sawmill to operate continually on timber from these stands.

PROCEDURES OF THE ERADICATION PROGRAM

General Procedures

Two ground surveys for oak wilt were made each year during the period 1954 through 1957. (During 1954, only one survey was made in the latter part of June.) The first survey was begun about the second week of June, and the second survey was usually begun the


BOUNDARY OF FOREST AREA
PORTION OF FOREST WHICH SERVED AS NONTREATED AREAS (180 ACRES)
EACH SQUARE EQUALS 40 ACRES
BASIC DATA FROM AERIAL PHOTOGRAPHS TAKEN IN 1939
DEPARTMENT OF FORESTRY
UNIVERSITY OF ILLINOIS

Map of Sinnissippi Forest, Oregon, Illinois. (Fig. 3)

second week of August. Each ground survey was made by walking straight lines at intervals of only a few chains, and each 40-acre compartment was scouted individually. After each infected tree was located, the stems of the trees were marked using a tree-marking gun. Since 1951, all infected trees were marked to designate the year each tree first wilted.

In 1957 and 1958 the ground surveys were supplemented by two aerial surveys each year. After comparing results from two years' data of ground and aerial surveys, the aerial survey was believed to be more efficient than the ground survey in locating diseased trees. Consequently, in 1959 and 1960 the biannual survey was made only from the air. A high-winged monoplane (Cessna 170) was used to make each aerial survey. Aerial surveying was accomplished by circling each individual compartment or group of compartments at an altitude of approximately 300 to 500 feet. In 1957, the entire forest was also photographed at an altitude of 2,000 feet and at close intervals in order to obtain overlapping of the frames. Kodachrome $2\frac{1}{4} \times 2\frac{1}{4}$ inch film was used to determine if infected trees showing wilt symptoms would be sufficiently visible on color film to make aerial photography a practical procedure. This supplementary method did not prove satisfactory and was not used in succeeding years. Although the majority of infected trees located by ground and aerial surveys were visible, and could be located by the aerial photography survey, the trees with small crowns and understory trees were not visible on color film at the altitude chosen for the test.

All trees having atypical wilt symptoms were sampled so that infection by the oak-wilt fungus might be confirmed by laboratory tests. Laboratory testing was done more often on trees of the white oak group, since both bur and white oaks showed considerable variation in symptom expression. In treated areas poison barriers were placed around all trees of the red oak group that were confirmed as being infected, but not around trees of the white oak group. Nevetheless, all diseased trees of the white oak group were poisoned. The poison barriers were established by poisoning all healthy oak trees of the red oak group which were within root-grafting distance of currently infected trees. Several pathologists have arbitrarily suggested that all healthy trees within 50 feet of a diseased tree should be poisoned, but the establishment of a 50-foot poison barrier around all currently infected trees was considered too costly an operation in a forested area the size of Sinnissippi Forest. Therefore, it was necessary to reduce the radius of the poison barrier in order to decrease the cost of the operation.

Table 1. — Basic Reference Table Used in the Eradication Program at Sinnissippi Forest to Determine the Maximum Root-Grafting Distances for Establishing Poison Barriers

Dbh of diseased tree plus dbh of healthy tree in inches	Maximum grafting distance in feet	Dbh of diseased tree plus dbh of healthy tree in inches	Maximum grafting distance in feet
2	12	26	38
4	13	28	41
6	15	30	43
8	16	32	45
10	18	34	48
12	21	36	50
14	23	38	52
16	26	40	54
18	28	42	56
20	31	44	58
22	34	46	60
24	36	48	62

A graph was drawn that tentatively related the dbh (diameter at breast height, measured in inches 4.5 feet above the ground level) of a diseased tree and the expected maximum distance that it could be root-grafted to another tree of any dbh. This graph was based on previous observations in Sinnissippi Forest that indicated the distance between wilt-killed and currently infected trees and their respective diameters. (Currently infected trees are those trees that express wilt symptoms the same year that control measures are being applied.) On the basis of this graph, a table which contained the essential information, in a form that could be easily referred to, was prepared (Table 1). This table was used as an aid in determining which trees should be poisoned. It should be emphasized that the root-grafting distances listed on this table were strictly followed throughout the six years of the control program in all timber and brush areas that were treated. Also, poison barriers were established only around currently infected trees. The table filled the need for standardizing an operation that could be followed for a number of years without destroying an excessive amount of timber.

All tree diameters were measured to the nearest inch with a diameter tape. Distances between trees were paced except where the distance, according to the graph, was very close to the maximum grafting distance for the two trees. In such cases the exact distance was measured with a steel tape.

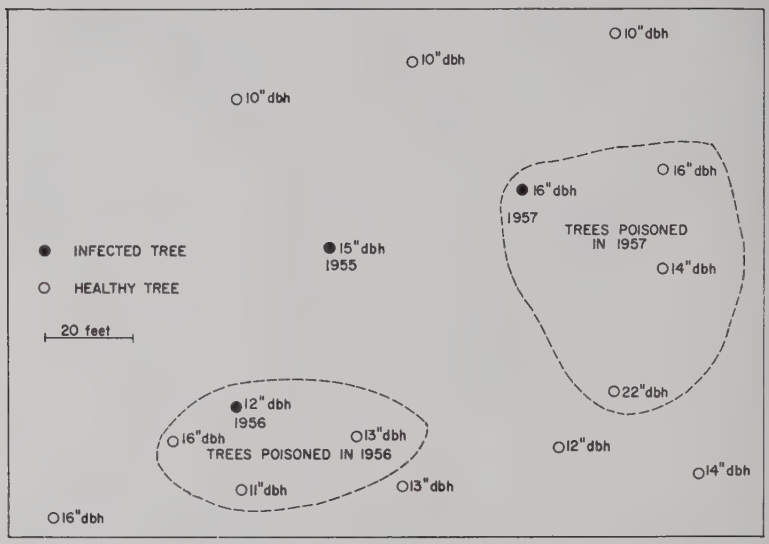


Illustration of a typical infection center and its treatment by the procedures used in the eradication program. (Fig. 4)

An example of a typical infection center[1] and the method applied to prevent further spread of the oak wilt fungus through root grafts by poisoning healthy trees is illustrated in Fig. 4. The blackened dots represent diseased trees that were infected and expressed wilt symptoms the year noted. The circles represent oak trees that were assumed to be healthy. The infection center was first treated in 1956 with the establishment of a poison barrier around one tree that wilted in 1956. Based on Table 1, three healthy trees were within grafting distance of the infected tree and were poisoned. In 1957 another tree became infected in the same center, and three more healthy trees were poisoned. Two of the trees that were within 50 feet of the tree which wilted in 1957 were not poisoned, since according to the reference table, Table 1, they were beyond root-grafting distance of the infected tree. In some instances it may be necessary to poison healthy trees beyond 50 feet if

[1] Infection center may be defined as two or more diseased trees of the red oak group that are oriented in such a manner that each tree is within root-grafting distance of another diseased tree. For practical purposes 50 feet is usually considered by most investigators as a maximum grafting distance between oak trees.

the combined diameters of the infected tree and the healthy tree are greater than 36 inches.

Barriers were not established around trees which were killed prior to the start of the eradication program in the major portion of the forest. This made it necessary to re-treat some of the centers more than once, since some trees surrounding infected trees became infected two or three years later. Poison barriers were established around all infected trees of the red oak group. They were not established around trees of the white oak group. Single-tree infection centers were treated in the same manner as described above. A single infected tree may be defined as an infected tree 50 feet or more from any other diseased tree. As it is used in this study, however, infected trees of small diameters (1 to 10 inches dbh) may still be considered as single-tree infection centers, even though the distance to other diseased trees of comparable size may be less than 50 feet.

Control of Oak Wilt in Reproduction Areas

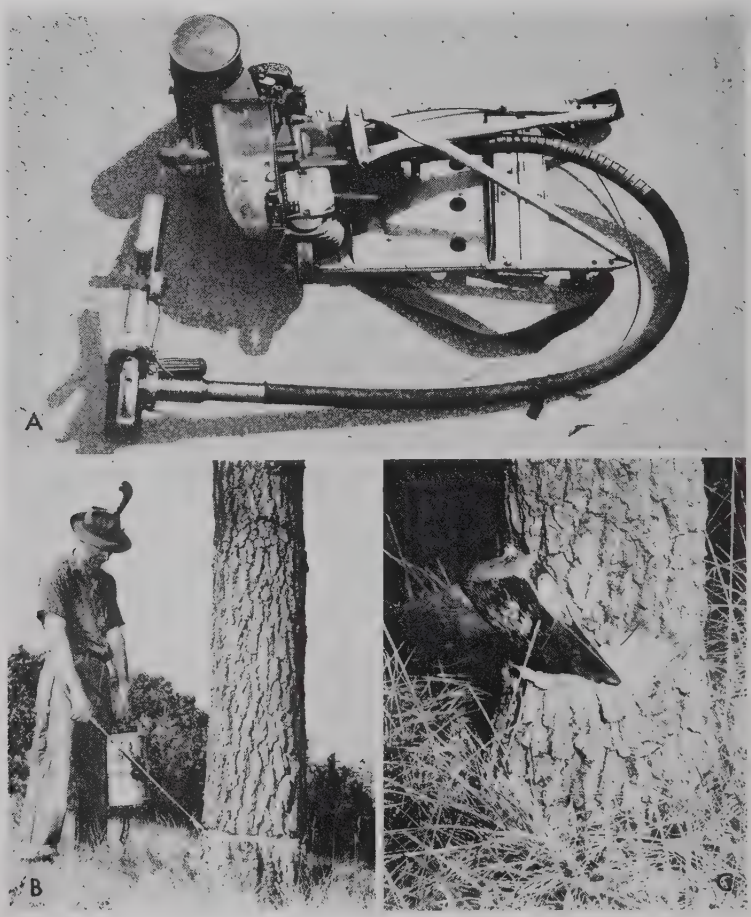
Reproduction areas referred to in this work are areas of small oaks which range in size from ¼ to 4 inches in diameter. Controlling the spread of the oak wilt disease in reproduction areas was one of the most difficult problems encountered in the eradication program. The numerous, scattered reproduction areas were, in most instances, dense and often covered 10 to 20 acres. When the oak wilt disease became established in large reproduction areas, it usually broke out in several isolated spots that were very difficult to relocate by the treating crew after they were found by the survey crew.

From experience and results obtained the first year of the program, a system evolved which made the work of the poisoning crew systematic and thorough in large reproduction areas. The general outline of each area was first rapidly walked by the man in charge of the treating crew. After the location of all of the infected trees was established, the larger healthy trees occurring 12 to 16 feet beyond infected trees were marked with paint for reference in identifying trees in the poison barrier. A second man delimited the outer extremity of the barrier by running string completely around all trees that had been marked by the first man.

The poisoning distance around reproduction areas varied as shown in Table 1. Where large infected trees occurred, the barrier was extended a greater distance than for small trees. All healthy trees occurring inside the string were then poisoned by the treating crew.

Chemicals Tested, Concentrations Used, and Methods of Application

In general the herbicides were applied in ax frills (Fig. 5C), made as close as possible to the ground level. However, another method of making frills was tried in which a gasoline powered machine was tested (Fig. 5A). After using the machine one summer, it was believed



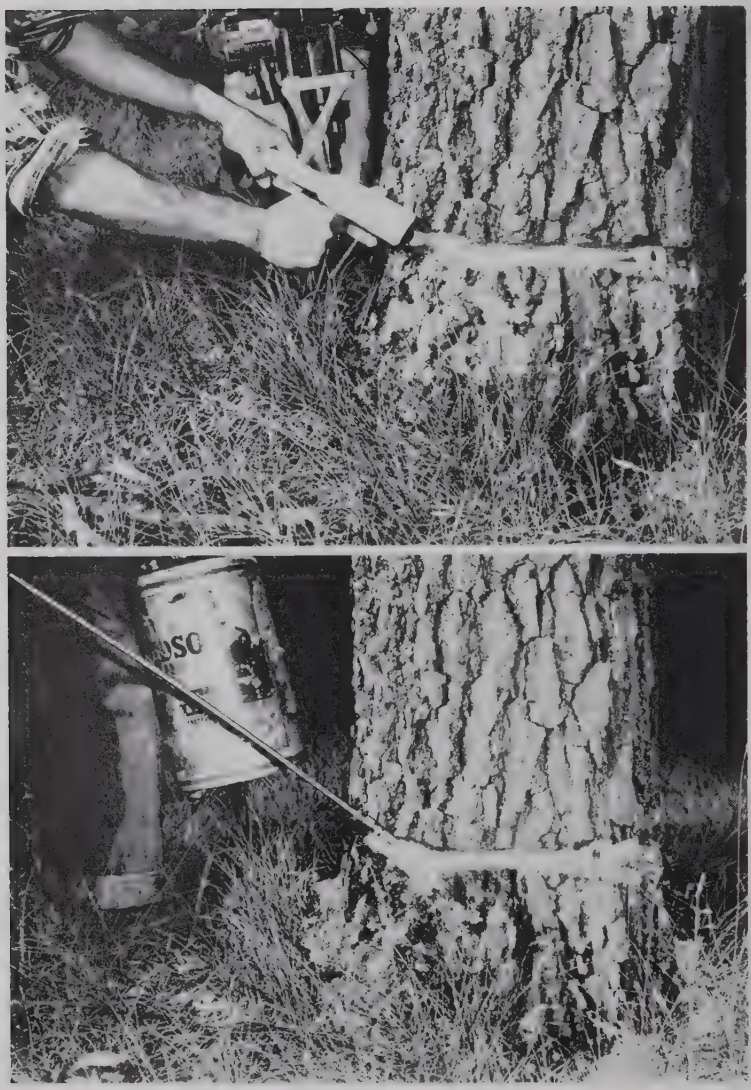
Gasoline powered machine called the Little Beaver. (Fig. 5A)
Method of applying liquid herbicides in frills. (Fig. 5B)
Close up of a typical ax frill. (Fig. 5C)

that the disadvantages outweighed the advantages. A major disadvantage was that the type of frill made by the cutting head of the machine (Fig. 6) did not hold a sufficient amount of liquid chemical for rapid uptake by the vessels of the wood.

In 1954 a water-suspendible formulation of 2,4,5-T was applied as a basal spray, in frills, and as a foliage application on small oak trees. The particular product used contained 4 pounds of acid equivalent per gallon of concentrate. Because satisfactory kill was not obtained with this type of 2,4,5-T, an oil-soluble 2,4,5-T was used in succeeding years. The oil-soluble 2,4,5-T was used at a concentration of 16 pounds acid equivalent per 100 gallons of fuel oil. Premixing large quantities of the herbicide with fuel oil was not satisfactory because the herbicide concentrate tended to separate and settle. Consequently, to obtain a thorough mixture of the herbicide and fuel oil, the liquids were mixed in the hand sprayers used to apply the chemical. The spray was mixed in a ratio of 1½ cups of 2,4,5-T concentrate in 2 gallons of fuel oil. Trees smaller than 1 inch dbh were usually poisoned by foliage application. Small trees 1 inch to 4 inches dbh were poisoned with 2,4,5-T applied as a basal spray. The spray was applied to the point of runoff and up to a height of 2 feet on the trunks. All trees 4 inches dbh and larger were frilled and the poison was applied in the frills.

Sodium arsenite was tested on a limited scale in 1956 to poison both healthy and infected trees. Because of the rapid kill obtained, sodium arsenite was used in the major portion of the forest in 1957 through 1960 to replace 2,4,5-T. In the last four years of the program, the use of 2,4,5-T was continued only in those areas where there was danger to humans and in reproduction areas. Sodium arsenite, under the brand name Atlas "D" (Debarking Compound), contains 42.5 percent active sodium arsenite, which is equivalent to 29.30 percent As_2O_3 or 4.0 pounds of As_2O_3 per gallon. Atlas "D" also contains a blue dye and a deer repellent. Sodium arsenite was applied in ax frills. A Hudson sprayer with the spray nozzle removed from the spray tube was used to dispense the chemical. The person dispensing the chemical circled the tree twice, allowing the chemical to run slowly into the frill (Fig. 5B). Very little runoff resulted when low pressure and careful dispensing of the chemical were used.

CMU, chemically (3,4-dichlorophenyl)-1, 1-dimethylurea, was supplied under the brand name Telvar. CMU at ⅛ pound (80 percent active ingredient) wettable powder per gallon of water was applied in a circle 6 feet in diameter around the base of each treated tree. Periodic shaking of the hand sprayer was necessary to keep the chemical in



Method of using the Little Beaver and a typical girdle made by the cutting head of the machine. (Fig. 6, upper)

Application of a liquid herbicide in a girdle made by the Little Beaver showing rapid runoff of the liquid. (Fig. 6, lower)

suspension. Ammate in a crystalline form was applied to frills made at or near the ground level. Approximately one cup of the chemical was applied to a tree 12 inches dbh.

Ammate and CMU were used in a few areas within the forest, but only on a limited test basis. Ammate was applied only in ax frills, while CMU was applied in ax frills and also as a soil application.

To reduce the fungus inoculum potential for overland spread, merchantable wilt-infected trees were cut and removed within two to three weeks after the second oak wilt control work was completed in August. All merchantable trees poisoned in establishing barriers were also salvaged at the same time wilt-infected trees were being removed. For the first four years of the program, all branches and trunk portions of merchantable wilt-infected trees 4 inches in diameter and larger remaining in the forest were sprayed with creosote. Nonmerchantable wilt-infected trees 3.6 inches or greater in dbh were cut and sprayed. In the last three years, the use of creosote was discontinued when it was found that sodium arsenite effectively prevented mat formation and limited bark-beetle breeding in diseased trees.

RESULTS OF THE OAK WILT ERADICATION PROGRAM

Proximity of Currently Infected Trees to Others Previously Killed by the Disease

Where oak wilt infection areas occur, it is impossible to know how much short-distance spread is represented by root-graft transmission. To understand more about the natural spread of the disease, it was necessary to very carefully observe and collect data for a number of years. During the seven-year eradication program, all infection centers, treated centers, or centers included in nontreated areas were visited one or more times. Data were recorded on all infection centers that had occurred since 1945. This made it possible to know the exact number of years that elapsed between the year each former wilt-killed tree was killed and the year new infection appeared in old infection centers. A careful record was kept on the diameters of trees, the distances between wilt-infected trees, and the date each tree wilted. These composite data recorded the complete history of all infection centers that were established during the period 1954 through 1960.

The relation of the number of newly infected trees and their distance from old wilt-killed trees is shown in Table 2. To establish separate infection centers, an arbitrary distance of 50 feet was con-

Table 2. — Effect of Distance Between Wilting and Wilt-Killed Trees

Distance in feet between current and old wilt	Number of infected trees	Percent of total observations
0–10	112	31.1
11–20	148	41.1
21–30	69	19.2
31–40	26	7.2
41–50	5	1.4
Total	360	100.0

sidered the maximum root-grafting distance for any size of tree. Data from trees within 50 feet of former wilt-killed trees are included. Isolated, single-tree infection centers occurring more than 50 feet from other wilt-killed trees are not included and were considered as infection centers arising from overland spread of the disease. It should be pointed out that the trees considered in Table 2 could have become infected either through root grafts or by short overland transmission, or, possibly more rarely, by both methods. Approximately 91 percent of the observed currently infected trees were within 30 feet of trees that died as a result of oak wilt, and 9 percent were within 31 to 50 feet of wilt-killed trees. These data indicate that a considerable amount of timber would be killed unnecessarily by establishing a 50-foot poison barrier around all currently wilt-killed trees.

The density of the oak stand and the diameter class were considered the two most important factors affecting the frequency of the new trees becoming infected through the root grafts. Density of the oak stand obviously should affect the frequency of root-grafting between individual trees. Also, as trees mature, the lateral roots extend a greater distance from their trunks, a factor which may also influence the frequency of root grafting.

The relation of the tree diameters to the distance between wilting and wilt-killed trees is illustrated in Table 3. As the combined diameters of the diseased trees increase, the number of infections over greater grafting distances also increase up to the point where the distance between trees becomes a limiting factor and the number decreases. It may be noted that the highest percentage of infection (44 percent) occurred in the diameter class of 23 to 30 inches dbh. All wilt trees of the red oak group infected by root-graft spread in the period 1954 through 1960 had an average diameter of 12.2 inches dbh. Since 12.2 inches was the average diameter and twice 12.2 is 24.4

Table 3. — The Relation of Tree Diameters to the Distance Between Wilting and Wilt-Killed Trees

Dbh of wilting plus dbh of wilt-killed trees in inches[a]	Number of wilting and wilt-killed trees where grafting distance in feet was					Number of occurrences observed	Percent of total observations
	0–10	11–20	21–30	31–40	41–50		
4– 6	5	..	..	..	..	5	1.4
7–14	10	3	..	..	..	13	3.6
15–22	27	45	20	1	..	93	25.8
23–30	48	66	33	12	..	159	44.2
31–38	20	29	13	12	1	75	20.8
39–46	2	5	3	1	4	15	4.2
Total number of trees observed	112	148	69	26	5	360	100.0

[a] A single wilt-killed tree which was clearly within root-graft distance of the currently infected tree was considered as the probable tree from which the oak wilt fungus came. The table does not include cases where more than one wilt-killed tree or a stump may have been root-grafted to the wilting tree.

inches, it would be expected that the combined diameter class of 23 to 30 inches would contain the greatest percentage of trees. A considerable decrease in the amount of root-graft spread occurred as the combined diameter of the two wilt trees extended beyond 30 inches. It would seem probable that as trees grow to almost mature size of 19 or 20 inches dbh, their lateral roots could be grafted considerable distances from the trunk. However, lateral roots may not grow radially as fast or as far in the larger trees as they become older and begin to reach a mature age.

Possibly a more logical explanation is that there were far fewer instances where the sum of two tree diameters would be more than 30 inches. In fact, Table 3 may be indicative of the relative frequency of trees of different diameters being within root-grafting distance of each other.

Activity of Oak Wilt Centers

The knowledge of natural cessation of oak wilt spread in established infection centers is important in a forested area where an experimental eradication program is being conducted. At the beginning of this experiment, the percentage of the oak wilt infection centers that would die out naturally was not known. Many infection centers in Sinnissippi Forest cover several acres and have been active for many years. Dying of oak trees in certain specific areas of the forest has been observed for 20 years by at least two individuals employed at the

Table 4. — Number of Years Infection Centers Remained Active Based on the Number of Trees in the Center

Number of diseased trees in infection centers	Number of infection centers active for[a]						Number of active centers observed	Percent of total infection centers observed
	1 year	2 years	3 years	4 years	5 years	6–13 years		
1	93[b]	..	..	..	..	..	93	33.3
2	..	30	2	4	..	..	36	12.9
3	..	18	1	3	3	..	25	9.0
4	..	9	1	2	1	1	14	5.0
5	..	5	1	..	..	1	7	2.5
6–10	..	..	2	3	9	34	48	17.2
11–20	..	..	..	3	2	32	37	13.3
21–30	..	..	..	..	..	10	10	3.6
31–100	..	..	..	..	..	9	9	3.2
Total number of centers	93	62	7	15	15	87	279	
Percent of total infection centers	33.3	22.2	2.5	5.4	5.4	31.2		

[a] Centers which were active 1 to 5 years were active sometime during the period of 1945 through 1954 and were inactive without any treatment from 1955 through 1960. Centers which remained active 6 to 13 years occurred in the nontreated portions of the forest from 1945 through 1958.

[b] Represents single tree infection centers which did not spread in six years of observation.

forest. Observations indicate that some infection centers tended to remain active year after year, but the spread in many other centers stopped after a year or two.

In the period 1945 through 1958, 279 separate infection centers were observed in nontreated areas which contained one or more infected trees 4 inches dbh or larger (Table 4). Ninety-three of the 279 active centers (33.3 percent) were single tree infections and did not repeat. Of 175 centers that contained no more than five trees each, only two centers (1.1 percent) were active after five years. Conversely, of 104 infection centers that contained six or more wilt-killed trees, 85 (81.7 percent) were active six years or more.

A large infection center may over a period of years die out and cease to have new infected trees. This may sometimes be due to the lack of healthy trees at its periphery. Bilbruck (1957) observed that the spread of the oak wilt fungus through the root system was slow. It is known, however, that the oak wilt fungus can live for at least three years in the roots of wilt-killed trees, Yount (1955). Boyce, Jr. (1960), showed that the distribution of the fungus within the roots varied considerably and in many cases was isolated several feet from the stem of an infected tree the same summer the tree became infected.

Table 5. — Relation Between the Time of Apparent Cessation of Oak Wilt Spread and the Appearance of New Wilt

Number of years elapsing	Number of infection centers	Percent of total infection centers observed
0[a]	12	4.8
1	59	23.4
2	61	24.2
3	54	21.4
4	31	12.3
5	28	11.1
6	7	2.8
Total	252	100.0

[a] Wilt-killed trees which were infected from a tree that wilted and died earlier the same summer.

An infection center may appear to die out for a period of time, only to break out again somewhere inside the infection center or at its margin. During a five-year period a record was made of the number of years elapsing between the apparent cessation of oak wilt spread and the appearance of new wilt in each infection center. These data, summarized in Table 5, indicate that the number of wilting trees is approximately the same in each of the established infection centers that were inactive for one, two, and three years. The number of new infections occurring in established centers is not significantly reduced until all wilt-killed trees in the infection area are at least five years old. Seven isolated infection centers which had no active wilt for six years became active again the seventh year.

Timber Loss Due to the Disease and to the Establishment of Poison Barriers

Mortality due to the oak wilt disease is very important in determining the effectiveness and economic practicability of an eradication program. Although 48 percent of the oaks in Sinnissippi Forest belong to the white oak group, and 52 percent to the red oak group, 93.4 percent of the trees killed or affected by the oak wilt disease were in the red oak group. The numbers of trees of each species are listed in Table 6. The greatest mortality occurred in the black oak species (member of the red oak group) in both the treated and nontreated areas of the forest. The loss in black oaks was larger than the combined mortality of all other species.

In establishing poison barriers around 724 infected trees in the treated portion of the forest, 2,075 healthy trees were poisoned in the

Table 6. — Mortality in Total Area of Forest Due to Oak Wilt, 1954-1960

Tree species	Average dbh	Number of trees	Percentage of total trees
Black oaks	11.5	780	66.8
Red oaks	14.9	254	21.8
Northern pin oaks	10.4	56	4.8
Shingle oaks	4.7	3	.3
White oaks	11.1	53	4.5
Bur oaks	12.1	21	1.8
Total		1,167	100.0

Table 7. — Healthy Trees Poisoned to Establish Barriers Around Currently Infected Trees of the Red Oak Group

Year	Number of infected trees	Merchantable, healthy trees poisoned		Number of trees poisoned per infected tree	Nonmerchantable, healthy trees poisoned		Number of trees poisoned per infected tree	All healthy trees, number of trees poisoned per infected tree
		Number	Average dbh		Number	Average dbh		
1954	61[a]	114	15.7	1.9	43	12.4	.7	2.6
1955	201	298	14.5	1.5	248	10.0	1.2	2.7
1956	183	326	14.5	1.8	146	9.1	.8	2.6
1957	79	170	15.1	2.2	97	9.6	1.2	3.4
1958[b]	77	168	14.2	2.2	78	9.7	1.0	3.2
1959[b]	75	169	14.4	2.3	7	9.1	1.0	3.2
1960[b]	78	175	14.1	2.2	65	8.7	.8	3.1
Total	724	1,354	14.6	1.9	721	9.8	1.0	2.9

[a] Less than one-sixth of the oak forest (227 acres of a total of 1,577 acres) had control measures applied in 1954.
[b] These data are the combination of data from treated and nontreated areas of the forest. Control measures were applied to the entire forest in 1958 through 1960.

five-year period (Table 7). Of the poisoned healthy trees, 65.3 percent were merchantable and 34.7 percent were nonmerchantable. (Estimation of merchantability was always made by the junior author in order to standardize the procedure and to eliminate any deviations that might occur if more than one man made the estimation.) An average of 1.9 merchantable and 1.0 nonmerchantable healthy trees were poisoned for every wilt tree.

In the total area of the forest, the average diameter of trees of the black oak species killed by the oak wilt disease was 11.5 inches and the average diameter for the red oak species was 14.9 inches. Black oaks occur predominantly on the poorer, dry, sandy soil. Red oaks are generally found on the better, more moist sites. The average annual oak wilt mortality, based on cubic-foot loss per acre, is given in Table 8. On a cubic-foot, per-acre basis, the greatest timber loss occurred on the good sites of the forest previous to the initiation of the eradication program. The loss on medium and poor sites was slightly less than the loss on good sites. After the establishment of a control program, volume

loss was reduced in the third year of the program, 1958, on good and medium sites in the treated areas, to less than one cubic foot per acre. The greatest reduction in the average loss on all sites also occurred this year, with a slight increase in 1959 and 1960 due to an increase in overland spread. The data also indicate a considerable decrease in loss in the nontreated area after the area was treated in 1958.

A compilation of the actual loss of merchantable timber due to the oak wilt disease and to establishment of poison barriers is presented in Tables 9 and 10. According to the forest inventory data compiled by Jokela and Lorenz (1957), the average annual production of sawtimber in the upland hardwood area of 1,577 acres was approximately 149 board feet per acre. The average annual mortality due to the oak wilt disease in the period 1945 through 1955 was 11.9 board feet per acre. This represents a loss of 8.0 percent of the total sawtimber production per year for a five-year period before the eradication program was started. This loss was reduced to an average of 2.5 percent of the total production in the latter three years of the program.

Table 8. — Volume Loss From Oak Wilt Infection of Trees 3.6 Inches DBH and Over

Site quality of timber	Total forest area, 1945–1955	Treated area of forest					Nontreated area of forest, 1956–1958, and treated, 1958–1960				
		1956	1957	1958	1959	1960	1956	1957	1958	1959	1960
	(average, cubic feet per acre, per year)										
Good	5.7	7.6	5.1	.8	1.8	2.8	8.6	...	8.7	...	...
Medium	3.2	2.8	1.3	.5	1.1	1.1	14.1	17.4	9.0	4.7	4.2
Poor	3.6	4.6	2.6	1.6	1.2	1.4	5.9	3.1	1.6	2.6	1.4
Reproduction	.8	.8	.3	.1	.6	.1	...	...	...	...	...
Average for all sites	3.1	3.5	1.8	.8	1.1	1.2	8.8	7.3	6.3	3.0	2.2

Table 9. — Average Annual Loss in Merchantable Timber From Oak Wilt Disease and From the Establishment of Poison Barriers; All Trees, Except Cull Trees, 9.6 Inches DBH and Over, Included

Site quality	Average[a] annual production	Average annual oak wilt mortality 1951–1955	Loss in treated area of forest									
			Oak wilt					Poison barriers				
			1956	1957	1958	1959	1960	1956	1957	1958	1959	1960
	(board feet, per acre)											
Good	246	19.6	27.5	18.7	2.9	6.5	10.1	144.7	112.6	19.9	44.8	69.7
Medium	191	9.8	9.3	4.2	1.5	3.3	3.2	28.7	12.3	5.8	12.8	13.6
Poor	143	10.5	14.2	8.0	6.9	5.2	6.0	21.4	17.4	12.3	9.2	10.8
Reproduction	54	2.4	2.6	.7	...	...	....	.3	....	.8	....	....
Average for all sites	149	11.9	10.6	5.7	2.9	4.0	4.4	25.5	17.1	7.3	10.0	11.0
Percent of total production		8.0	7.1	3.8	1.9	2.7	3.0	17.1	11.5	4.9	6.7	7.4

[a] Based on 1949 and 1954 forest inventory data compiled by Jokela and Lorenz (1957).

Table 10. — Volume of Merchantable Oak Timber Lost Due to the Oak Wilt Disease and to the Establishment of Poison Barriers; All Trees, Except Cull Trees, 9.6 Inches DBH and Over, Included

	Year	Oak wilt mortality, board feet, per acre		Total mortality in board feet (1,648 acres)	Loss in board feet from poison barriers		
		Area first treated in 1958 (180 acres)	Area first treated in 1955 (1,468 acres)		Total acreage	Total board feet	Board feet, per acre
Nontreated	1945 50.....	33.9	42.2	67,996	.....	.....	....
	1951.....	42.4	17.5	33,278	.....	.....	....
	1952.....	13.1	5.6	10,657	.....	.....	....
	1953.....	13.1	8.7	15,066	.....	.....	....
	1954.....	18.3	11.0	19,435	.....	.....	....
	1955.....	25.5	10.4	19,932	1,468	49,030	33.4
	1956.....	27.3	10.6	20,425	1,468	40,027	27.3
	1957.....	24.2	5.7	12,749	1,468	27,025	18.4
Treated	1958.....	14.6	2.9	6,946	1,648	16,963	10.3
	1959.....	9.5	4.0	7,649	1,648	15,770	9.6
	1960.....	7.0	4.4	7,732	1,648	17,347	10.5
Total board feet..				221,865		166,162	

With the establishment of poison barriers, an extremely heavy cut of merchantable timber occurred, particularly in the good sites. However, each year the loss was reduced. In 1958, the third year of the program, the total volume of merchantable timber of diseased and poisoned healthy trees was reduced to below the average annual loss from oak wilt without the control program. The total amount of timber loss increased slightly above the average annual loss in both 1959 and 1960; again this was due to an increase in the amount of overland spread of the disease.

Jokela and Lorenz (1957) estimated the total mortality on a per-acre basis for each timber site in Sinnissippi Forest. Total mortality includes the loss from fire, insects, windstorms, tree diseases, suppression, and all other causes. A comparison of the oak wilt mortality and the total mortality was made by the writers (Table 11). This comparison shows that during an 11-year period, 45.6 percent of the total mortality was attributable to the oak wilt disease. The data also show that the percentage of oak wilt mortality in the good sites was relatively low when compared with the timber loss from other causes. The high mortality in the good sites from causes other than oak wilt is due to the fact that the good sites represent mature timber in which suppression, wind damage, and natural decline are quite high.

Table 11. — Comparison of Total Mortality With Oak Wilt Mortality; All Trees 3.6 Inches DBH and Over, Included

Site quality	Total[a] mortality, average cubic feet, per acre, per year	Oak wilt mortality, 1945–1955	
		Average cubic feet, per acre, per year	Percent of total mortality
Good	19.8	5.7	28.8
Medium	7.2	3.2	44.4
Poor	6.7	3.6	53.7
Reproduction	1.0	.8	80.0
Average for all sites	6.8	3.1	45.6

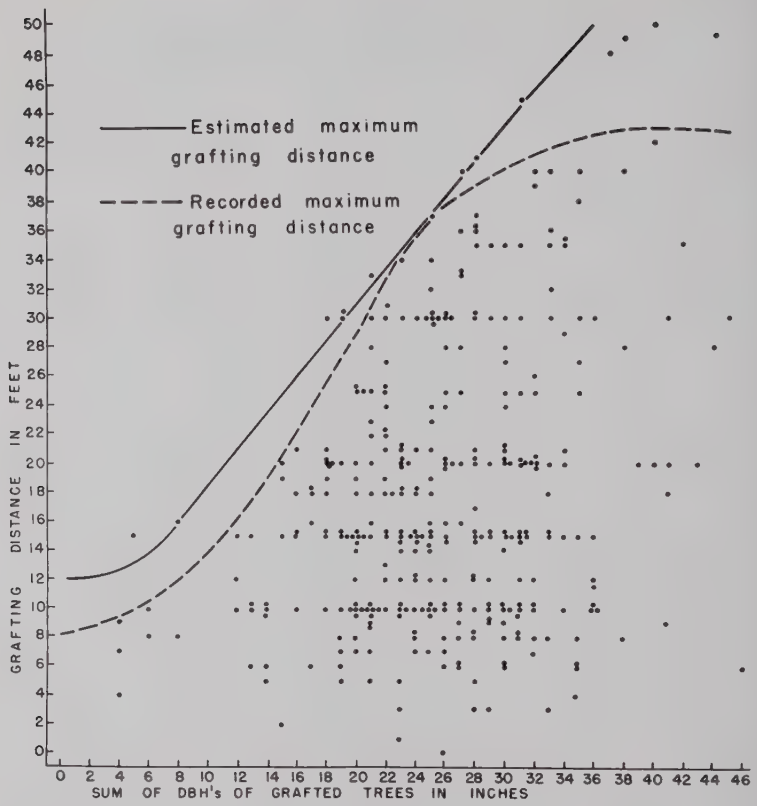
[a] The figures are based on 1949 and 1954 forest inventory data compiled by Jokela and Lorenz (1957).

Effectiveness of the Poison Barriers

The establishment of poison barriers around currently infected trees has proved very effective in preventing further root-graft spread of the oak wilt fungus in Sinnissippi Forest. In the five-year program, barriers were placed around 571 currently infected trees. In the entire program only 14 trees have become infected beyond the barrier zone. Because of their small diameter and distance from wilt-killed trees, it was assumed that four of the 14 trees became infected by overland spread. The remaining 10 trees, which it was thought were infected through root grafts, could have become infected before the poison barriers were established. Presumably, the fungus can pass from the unkilled portions of the roots of poisoned trees through graft unions into roots of adjacent, healthy trees.

The proximity of currently infected trees to wilt-killed trees is illustrated in Fig. 7. Each dot represents an infected tree close to only one other wilt tree. The diameters of the two trees were added and are plotted on the abscissa of the graph. The distance between the two wilt trees is indicated by the grafting distance represented on the ordinate of the graph. It should be noted that very few trees lie outside of the solid line, which represents the maximum distance the poison barrier was established beyond all currently infected trees. The dashed line represents the extremes of actual root-grafting distances related to the total diameters of the trees.

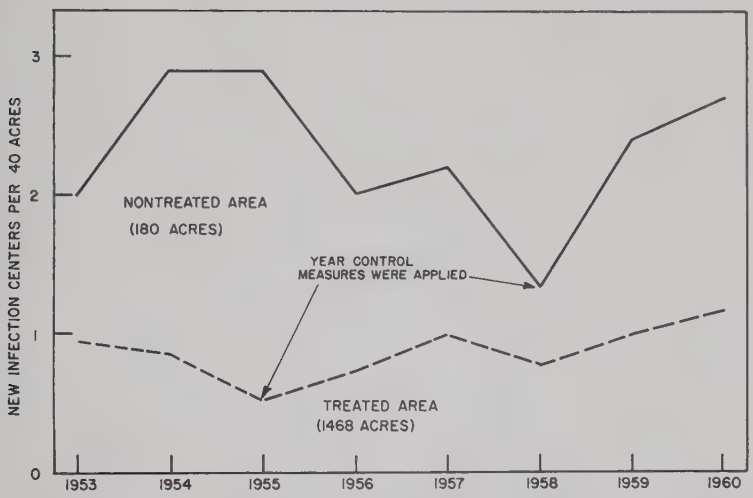
In the initial planning of the eradication program, 180 acres of the forest were set aside to serve as a nontreated area. This area, although small in relation to the total 1,648 acres of forest, contained a large number of active wilt areas. A few of the wilt areas in this 180 acres were quite large in size and in number of wilt trees. One complicating factor, which was realized in the beginning of the program, was that a


Estimated maximum grafting distance
Recorded maximum grafting distance
GRAFTING DISTANCE IN FEET
SUM OF DBH's OF GRAFTED TREES IN INCHES

Proximity of currently infected trees to others previously killed by oak wilt. The sum of the dbh of the currently infected tree and the dbh of the wilt-killed tree is plotted against the distance between the two. (Fig. 7)

constant source of inoculum would be present for overland spread from the nontreated portion into the treated portion of the forest. Experimental inoculation areas, which served as additional areas for inoculum, were also present in certain other parts of the forest. Inoculation areas and nontreated areas would not normally be present in a large-scale control program.

After three years of data were collected, the nontreated area was treated following a different procedure than was used in the beginning of the program on the larger portion of the forest. The procedure was essentially that of poisoning all healthy trees within 30 feet of infected trees that were dying or that had been killed in the previous three

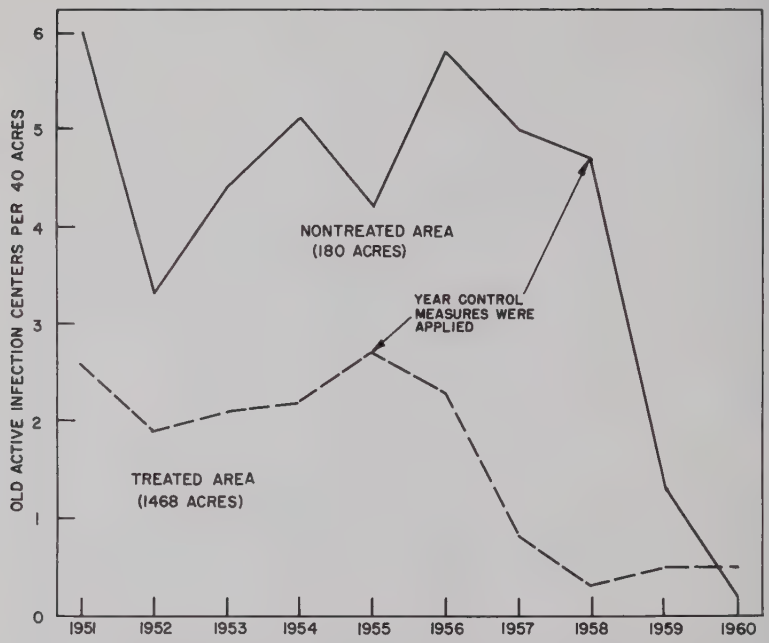


Average number of new infection centers per 40 acres in the period 1953-1960. Control measures applied in 1955 (treated area) and 1958 (nontreated area) were continued during succeeding years. (Fig. 8)

years. Following this treatment, which was first used in 1958, barriers were established only around currently infected trees in 1959 and 1960. The number of new infection centers in the treated area of the forest from 1953 to 1958 remained fairly constant, ranging from 0.9 to 0.5 per 40 acres of forest (Fig. 8). However, after 1958 there was an increase in the number of infections due to overland spread to 1.1 infections per 40 acres. In the nontreated area, new infection centers ranged from 2.9 per 40 acres in 1954 to 1.3 in 1958. Here the significant drop in new centers that occurred after 1955 may be attributed to the reduction of inoculum in the treated area which surrounded the nontreated portion. An attempt to eradicate the disease in the nontreated area brought about a definite increase in the number of new infection centers resulting from overland spread.

The average number of old active infection centers per 40 acres for the period 1953-1958 in the treated and nontreated portions of the forest is represented in Fig. 9. The actual effect of the eradication program is illustrated more clearly by the rather rapid decline in the number of old centers in the treated area after the initiation of the program in 1955 and in the nontreated area after control measures were applied in 1958.

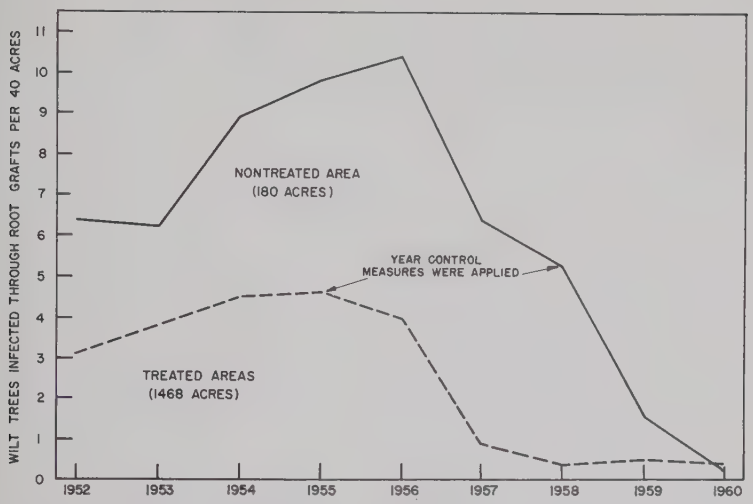
The average number of diseased trees per 40 acres which became infected in established oak wilt centers has shown a steady decline in



Average number of old infection centers per 40 acres in the period 1951-1960. After control measures were first applied (1955 and 1958 in treated and non-treated areas respectively) there is a steady decline in the number of old centers. (Fig. 9)

both areas after control measures were applied (Fig. 10). The more rapid decline in the smaller area, which was not treated until 1958, reflects the more drastic control measure of poisoning all healthy trees within 30 feet of infected trees that had been killed in the previous three years and around all currently infected trees. Essentially it took only two years to obtain control of root-graft spread by using the method of poisoning around trees killed in previous years, whereas control was not obtained until the third year, when barriers were placed only around currently infected trees.

As discussed under activity of oak wilt centers, the data show that a period of one to six or more years may elapse before a new infection appears in established oak wilt centers. Infection definitely appears to occur through root-graft unions with wilt-killed trees that have been dead for a number of years. This is a real problem when attempting to establish poison barriers around only currently infected trees. Table 12 adds further support to this fact. The infection centers which were



Average number of diseased trees per 40 acres infected through root graft spread in the period 1952-1960. (Fig. 10)

Table 12. — Number of Infection Centers on Which the Application of Control Measures Was Made One or More Times. Centers Listed Are Those in Which Poison Barriers Were Placed Only Around Currently Infected Trees During the Period 1954-1958

Number of trees in infection centers when last treated	Number of centers treated listed according to the number of times treated			Total number of centers treated
	1[a] treatment	2[a] treatments	3[b] treatments	
1	70	..	..	70
2	12	3	..	15
3	6	6	1	13
4	11	3	..	14
5	10	4	..	14
6–10	15	10	1	26
11–20	7	15	4	26
21–30	2	3	3	8
31–40	4	1	..	5
41–50	..	1	1	2
51–60	..	..	..	..
61–100	..	1	..	1
Total	137	47	10	194
Percentage of total	71	24	5	

[a] Centers which were treated in 1954-1956 and which have not been active since 1956.
[b] Number of centers which were treated in 1954-1958 and which have not been active since 1958. Some of these centers may become active again and it will be necessary to re-treat.

treated are listed according to the number of times an individual center had to be treated to arrest further spread. It will be noted that as the number of trees in the infection center becomes greater, the number of centers requiring additional treatments also increases.

Control of Oak Wilt in Reproduction Areas

The treatment of reproduction areas containing wilt was started in 1954 and extended through 1960. The numbers of reproduction areas treated during this period are given in Table 13. Retreatment of some of the areas was started the second year of the program. This was necessary because in some cases complete kill was not obtained in some of the healthy trees within the poison barrier. In other cases not all of the wilt-killed trees were seen in the heavy brush, and consequently the barrier had to be widened to include those new trees.

Of the many thousands of healthy trees occurring just outside of the poison barriers, only 10 trees became infected in reproduction areas. The main object of the first three years of the program was an attempt to contain oak wilt within a barrier of poisoned dead trees. Once definite barriers were established, the second objective was to poison all of the healthy and wilt-killed trees inside of the barriers with one treatment, provided the areas had a recurrence of infected trees. As the time required to treat the oak wilt areas in the forest became less, in 1957 and 1958, more time was available to poison those trees occurring inside the barriers. Some of the reproduction areas of infected trees were so large that it required a week or more for a crew member to complete the treatment of all oaks inside the barriers. Even though barriers were established around the large brush areas, oak wilt would have been present for many years inside the area if all of the healthy oaks were not poisoned to prevent further root-graft spread.

Control of Oak Wilt in the White Oak Group

Difficulties were experienced by the survey crew in detecting infected trees of the white oak group. This has, to a certain extent, limited the efficiency of the oak wilt survey program. Approximately

Table 13. — Number of Brush Areas With Active Oak Wilt in the Treated Portion of the Forest

Type of brush areas	1953	1954	1955	1956	1957	1958	1959	1960	Total
New	12	26	13	6	16	3	4	12	92
Re-treated	..	..	25	13	14	11	3	7	73
Total	12	26	38	19	30	14	7	19	165

Table 14. — Number of Oak Trees of Each Species Killed by the Oak Wilt Disease, 1953-1960

Species of oak	Number of trees	Percent
Red oak group		
Q. velutina	780	66.8
Q. rubra	254	21.8
Q. ellipsoidalis	56	4.8
White oak group		
Q. alba	53	4.5
Q. macrocarpa	21	1.8
Q. imbricaria	3	.3
Total	1,167	100.0

48 percent of the oak timber in Sinnissippi Forest is of the white oak group. However, in the final tabulation of the total number of oaks of each species killed by oak wilt, only 6.6 percent were of the white oak group (Table 14).

A wide range of variability in the symptoms of infected white oaks was accepted. In those few cases where the wilt symptoms were definitely atypical, branch samples were taken to isolate the oak wilt fungus. The recovery of the oak wilt fungus from those samples collected from trees expressing atypical symptoms was very high. Resampling was made of many white oaks which were not positively diagnosed from the first collection of branch samples, and many times the oak wilt fungus was recovered from the second sample.

An attempt was made to relocate those white oaks which had been marked as having oak wilt in earlier surveys before the eradication program was initiated. Circumstantial evidence indicates that a high percentage of the white oaks recovered after initial infection.

Fair but slow kill of white oaks was obtained by using Ammate and 2,4,5-T in frills. Often trees did not die for two or three months after they were poisoned. Sodium arsenite, on the other hand, gave very rapid and complete kill. Symptoms from arsenite poisoning usually would be expressed by the foliage in 24 to 48 hours. Treated trees were very often completely dead to the ground level in less than four days. Because sodium arsenite was a very effective chemical, it alone was used in 1957 through 1960.

Time and Labor Required for Control Work

During the early years of the survey at Sinnissippi Forest, ground crews walked each 40-acre compartment three times across, and this was done twice each year. The first survey was done in June, and the second in August. With two men on the ground survey, this required

each man to cover approximately a 200-foot strip each trip. The average man days required to complete a single survey during the years of 1955 to 1957 was 23 days for a coverage of 1,405 acres.

During the later years, aerial survey work replaced the ground crew work in all areas except reproduction areas where oak wilt had been active for many years. These areas were surveyed in August on the ground by the control crew as they were carrying out the control measures. A four-seated, high-wing Cessna with pilot was chartered for $14 per hour. Three men with maps recorded locations of trees exhibiting wilt symptoms as the plane circled over the forest.

Control work was generally done with a four-man crew. Principally two men did the frilling and applying of the herbicides, while the remaining two men measured and marked the trees and recorded the data. The total number of man days required for a crew to carry out the control measures is given in Table 15.

EXPERIMENTS RELATED TO POISONING OF OAK ROOTS

Chemical Application in Ax Frills

An experiment was conducted to determine if the oak wilt fungus in the roots of infected trees could be killed by chemical means and thus prevent the oak wilt fungus from passing through the root systems from one tree to another. In August 1954 wilting red oaks, *Quercus rubra,* and black oaks, *Q. velutina,* were treated with four fungicides to determine if downward translocation, in sufficient concentration to kill the oak wilt fungus in infected roots, would occur in

Table 15. — Labor Required for Control Work by Years

Year	Acres oak forest covered	Number of areas observed[b]	Total number of trees frilled and poisoned	Number of reproduction areas treated	Man days required
1954[a]	227	39	157	11	12
1955	1,405	117	546	38	72
1956	1,405	113	472	19	46
1957	1,405	64	356	30	38
1958	1,577	64	323	15	36
1959	1,577	74	315	7	26
1960	1,577	78	316	19	26

[a] Control work was done only once in 1954 instead of twice per summer as was done in the following years.

[b] This includes only areas in which work was done. There were a small number of other areas visited each year, which upon close examination were not considered to be oak wilt. Time involved in examining these areas is included in the total time involved in the control work.

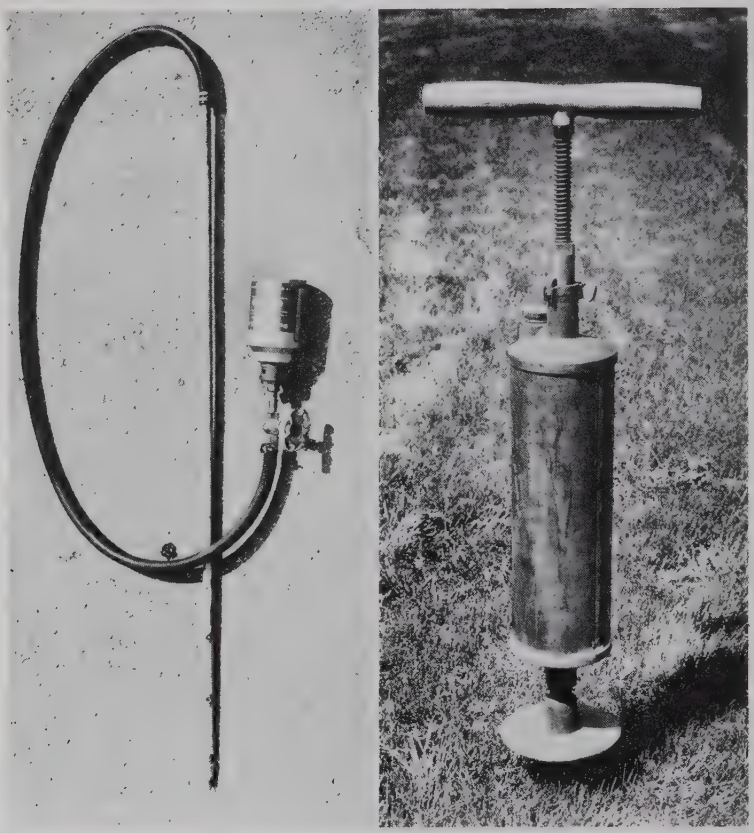
late summer. Phenylmercuric acetate (Tag 331), copper naphthenate, pentachlorophenol, and sodium pentachlorophenate were applied in basal ax frills. Four infected trees were treated with each chemical. Root samples were collected during October and November from each of the 16 treated trees and from 5 wilt-killed trees which had not received treatment. Root sections, 12 inches long, were taken from four sides and approximately 3 feet from the trunk of each tree. Attempts to isolate the oak wilt fungus were made from each root section.

Roots of trees treated with four herbicides used in the eradication program were examined to determine the amount of root kill resulting from the use of each poison. The four herbicides used were Ammate, 2,4,5-T, CMU, and sodium arsenite. In the summers of 1956, 1957, and 1958, observations were recorded on the amount of root-kill resulting from the use of these four herbicides applied in frills to both healthy and diseased trees. These observations were made one year after the trees had been poisoned. A few wilt-killed trees which had not been poisoned were also examined one year after they were killed to determine the extent of root-kill resulting from infection by the oak wilt fungus. In all cases the excavation and examination of the roots of poisoned trees was continued far enough from the stump to determine the average distance roots had been killed.

Chemical Application to the Soil

Six chemicals were tested on a small scale in 1954 and 1957 to determine if the roots of oak trees could be killed in a limited zone without causing injury to adjacent trees. The basic idea was to break root connections between oak trees. The chemicals tested were Vapam, methyl bromide, sodium pentachlorophenate, copper naphthenate, CMU (Telvar), and Monsanto soil sterilant, CP376. The chemicals were injected or applied to the soil surface. Methyl bromide (Dowfume MC-2) was injected 1½ feet into the soil with a soil needle. The needle and valve arrangement is shown in Fig. 11, left. The material was injected at one-foot intervals in a concentric ring 3 feet from the base of the tree. The material was applied at ½, 1, 1½, and 2 pounds per tree, and only one tree was treated at each quantity level. One-pound pressurized containers facilitated rapid and comparatively safe application of the methyl bromide.

Vapam (sodium N-methyl dithiocarbamate dihydrate) was injected into the soil in a liquid form using the Maclean's Fumigun with a 6-inch needle (Fig. 11, right). Concentrations of ¾ and 1 quart of the original stock solution (4 pounds per gallon of formulation) diluted in



(*Left*) Soil injection needle with attached pressurized can containing methyl bromide. (*Right*) Maclean's Fumigun. (Fig. 11)

3 quarts of water were used. Only one tree was treated with each concentration. The total volume of each concentration was injected at 6-inch intervals in a concentric ring 3 feet from the base of each tree.

The four chemicals, sodium pentachlorophenate, copper naphthenate, CMU (Telvar), and CP376 (orthonitrochlorobenzene) were applied with a Hudson hand sprayer in a band 6 inches wide and 4 feet from the base of each tree. Sodium pentachlorophenate was applied at rates of 7 and 12 ounces in 3 quarts of water, copper naphthenate at 2 ounces in 1 gallon of water, CP376 at 1 pint (4 pounds active ingredient per gallon) in 1½ quarts of water, and Telvar at ¼,

⅛, and 1/16 pound (80 percent active ingredient in wettable powder) in 1 gallon of water per tree. The chemicals, methyl bromide and Vapam, were tested on healthy black oak trees that ranged from 4 to 8 inches dbh. The chemicals, sodium pentachlorophenate, copper, naphthenate, CMU, and CP376, were tested on healthy black oak trees ranging from 5 to 13 inches dbh. Approximately 10 weeks after the chemicals were applied, an examination of the roots of all treated trees was made. All roots in the zone of application to a depth of 2½ to 3 feet were examined to determine if root-kill had been complete.

Results on Chemical Application in Frills

In preliminary root-poisoning experiments, Tag 331, copper naphthenate, pentachlorophenol, and sodium pentachlorophenate were tested by applying the chemicals in frills. The oak wilt fungus was isolated from roots of all 16 treated trees as well as from roots of five wilt-killed trees which received no treatment. Since there was no indication of downward movement by any of the four chemicals, testing of these chemicals was not continued.

There was no apparent difference in the extent of root-kill between poisoned-healthy and poisoned-infected trees with any of the chemicals tested. The data in Table 16 indicate that 2,4,5-T, Ammate, and CMU did not cause a significant increase in root-kill over the amount killed by the oak wilt fungus. The use of sodium arsenite in frills resulted in a significant increase in the number of trees showing root-kill. Roots were killed a greater distance from the stump by sodium arsenite than by the other herbicides. Sodium arsenite also increased the amount of root-kill on poisoned, infected trees compared to infected trees not poisoned.

In the last three years no mat formation was observed on diseased trees treated with sodium arsenite when the trees were in a comparatively early stage of wilt. Early detection and treatment of infected trees was possible by making two surveys during the summer. In addition to limiting mat formation, sodium arsenite has shown promising results in limiting bark borer breeding on the trunks of treated trees for a period of at least one year. The arsenite appears to be translocated in a concentration sufficiently high to poison most of the adult beetles and all of the larvae on the trunk to a height of 20 to 50 feet. Insect larvae were only found on a few of the smaller branches on many wilt-killed trees. These observations were made early in the year following the summer in which the trees were poisoned. Poisoned trees were often invaded by borers in the year following treatment.

Table 16. — Amount of Root-Kill on Trees Poisoned With Herbicides or Killed by the Oak Wilt Disease

Treatment	Total trees examined	Trees with no root-kill, number	Trees with some root-kill, number	Average distance roots were killed from stump in feet
2,4,5-T	31	26	5	1– 2
Ammate	8	6	2	1– 2
CMU	5	5	0	0
Sodium arsenite	40	7	33	5–10
Wilt-killed trees (no treatment)	32	20	12	1– 2

The prevention of bark beetle attack was considerably more effective on treated, healthy trees than on infected trees due to more rapid and complete uptake of the poison by healthy trees. Sodium arsenite also appeared to act as a repelling agent for borers for at least one year. This assumption is based on the fact that fewer borer holes and larval galleries were observed on trees treated with sodium arsenite than on trees treated with 2,4,5-T.

Results on Chemical Application to the Soil

Vapam and methyl bromide killed all roots up to 3 inches in diameter and to a depth of 3 feet. The chemicals, sodium pentachlorophenate, copper naphthenate, CMU, and CP376, failed to kill any roots in the zone of application. Some chemical injury was observed on two of four trees treated with Vapam. Methyl bromide killed one of four treated trees. No chemical injury from Vapam or methyl bromide was observed on adjacent healthy trees, although some small trees 4 to 6 inches dbh were within 10 to 15 feet of the poison zone.

Jones and Bretz (1958) reported that sprouting of poisoned trees was relatively common. Very few cases of sprouting were observed on trees poisoned with the four herbicides used in Sinnissippi Forest. One major reason for the small amount of sprouting may be that the ax frills were made close to the ground level.

CONCLUSIONS AND RECOMMENDATIONS

Observations and data reported here provide information on the effectiveness of control measures that were employed under conditions existing in northern Illinois. Sinnissippi Forest was chosen because the oak wilt disease was well established in both large timber and in brush areas and because the forest represents a variety of sites, forest

types, and age classes. A control program was justifiable, since the area contains native timber bearing a stand of almost 8 million board feet, 90 percent of which is oak. Because the forest was organized for management and research, it was possible to carry on an intensive control program which conformed with established forest practices.

The perpetuation of the oak wilt fungus is not necessarily dependent upon fruiting mats to survive long periods of adverse conditions, but appears to depend upon continued existence of the fungus in an oak wilt area where new inocula are constantly being produced. Although the authors recognize the importance of fungus mats in relation to overland spread of the disease, they wish to emphasize that the inoculum potential for spread to new areas is present as long as there are active infection centers. The authors believe that the first objective in eradicating oak wilt should be to prevent further local spread through root grafts to surrounding healthy trees. The second objective should be to prevent overland spread by destroying the inoculum potential in aboveground parts of infected trees.

Although suspected vectors of the oak wilt fungus have been reported to be inefficient, the fungus continues to spread long distances. Overland spread of oak wilt in Sinnissippi Forest has increased slightly in both areas under observation, although the number of trees infected through grafted roots decreased. Only seven trees were observed on which fungus mats were formed in the past three years. This fact suggests that there are sources of inoculum other than fungus mats.

A continuous survey and eradication program appears to be essential to prevent local spread of the fungus. Elimination of perpetual inoculum in a large forested area is largely dependent upon an effective and complete survey to locate all infected trees. Of all problems encountered in carrying out an effective control program in Sinnissippi Forest, locating active wilt areas was the most important and difficult to overcome. Effectiveness of the survey was increased to a great extent by supplementing the ground survey with an aerial survey. Both types of surveys were made twice each year, but this coverage was not completely effective in locating all infected trees. Aerial coverage of the forest would possibly have been more satisfactory if a helicopter instead of the conventional, high-winged monoplane had been employed.

Data on proximity of diseased trees indicate that 72 percent of currently infected trees observed in Sinnissippi Forest were within 20 feet of trees killed by oak wilt and 91 percent were within 30 feet (Table 2). This supports the authors' earlier contention that a 50-foot barrier around all infected trees, regardless of size, would necessitate killing many healthy trees, only a few of which would become infected

through root-graft spread. In addition to unnecessarily killing large numbers of trees, the 50-foot barrier also involves great expense.

With no control many infection centers may "die out" over a period of years, and others may continue to have active wilt each year until large areas of timber are killed. In a few cases single-tree infection centers within a few hundred feet of each other enlarged so that two or more original centers coalesced into one. Both overland and root-graft spread of the fungus occurred, and finally very few living trees remained in the centers. Fortunately, many single-tree infection centers did not spread. Only 1 percent of all centers which contained five trees or less were active after five years. Eighty-two percent of those centers which contained six or more trees remained active for several years.

An intensive study of active infection centers indicated that there were varying periods of cessation of further root-graft spread of the fungus. The periods of inactivity varied from one to six years. Unless poison barriers are established around every wilt-killed tree, complete prevention of further root-graft spread cannot be assured. Therefore, if poison barriers are established only around currently infected trees, it would be necessary to examine the centers annually for at least three years to determine if further spread occurs. In Sinnissippi Forest 71 percent of 194 treated centers required only one treatment to arrest further root-graft spread. Twenty-four percent required two treatments, and 5 percent required three treatments. Re-treating an area several times may be considered as a rather inefficient control. However, the majority of centers which required more than one treatment contained 6 to 100 wilt-killed trees. If barriers had been placed around all large infection centers, it would have been necessary to poison a much greater number of trees to prevent the possibility of only a few trees becoming infected. It would seem that such a procedure is only warranted in forested areas which are difficult to reach or in timbered areas that are low in value. Establishment of poison barriers around all wilt-killed trees occurring in valuable timber is almost prohibitive unless the timber can be readily harvested. Data presented in Table 10 indicate that the amount of merchantable timber loss from establishment of poison barriers was greatest on the good forest sites. Also, the heaviest loss from the establishment of the poison barriers in the good sites at the beginning of the second year (1957) was about five times the average loss from oak wilt. Timber loss from establishment of poison barriers in all timber sites appeared to drop off rather abruptly after the second year but increased slightly the fourth and fifth years. At the end of the third year of the control program, the total loss of timber from both wilt-killed and poisoned trees was a little less than the

loss that might be expected from natural spread of oak wilt had there been no control. The amount of timber loss in the last two years of the program (1959 and 1960) increased above the annual average loss from natural spread. The increase was brought about by an increase in overland spread of the disease.

Data obtained from four years of observations (1954 through 1957) showed that root-graft spread of the disease could be controlled by the poison barrier method, and it also showed that overland spread of the disease was not reduced but did show a slight increase in the treated area. Therefore, the nontreated area was treated in 1958 using different procedures to determine if overland spread could be controlled. By placing barriers around all infected trees that were dying and around those that had been killed in the previous three years, an extremely heavy cut of timber was made, even though the barrier was only 30 feet in radius around each oak wilt tree. This method of treatment gave excellent results in controlling root-graft spread by bringing about a very rapid reduction in the number of old active infection centers. Only six infection centers that had not been treated the first year had to be treated the second year, and it was necessary to treat only one center the third year. Again, overland spread of the disease does not appear to be controlled, since the number of trees recorded remained quite close to the average number recorded before the control measures were applied.

An essential part of the control program at Sinnissippi Forest was the establishment of poison barriers at varying distances based on diameters of diseased trees. The table used to standardize the distance of establishing the poison barriers around currently infected trees, Table 1, was not changed throughout the program. However, differences in oak species, soil types, and climatic conditions may require an increase in the distances necessary to prevent root-graft spread. It is doubtful whether the distances may be shortened and still be effective. There is an indication, however, that the barrier should be widened in timber that ranges from 11 to 13 inches in diameter. It was in this size of timber that the actual distance of root-graft spread was very close to the maximum grafting distance estimated at the beginning of the program. A factor that cannot be ignored in interpreting this data is that some trees could have become infected by short overland spread, although very few trees which became infected by overland spread occurred near the outer periphery of the poison barrier.

Establishment of poison barriers around infection centers in brush was very time-consuming. Generally, large areas had to be re-treated two or more times. In most cases several hundred healthy trees were

left inside the poison barriers, and oak wilt continued to spread. It was then necessary to poison all living trees within the barriers.

Only 6.6 percent of the total number of infected trees were of the white oak group. Very few instances of short overland or root-graft spread were observed from infected bur or white oak trees. Consequently, it was not believed practical to place poison barriers around either species. Locating and identifying infected trees of the white oak group is a difficult problem in an eradication program. Frequently only a few branches die each year, and infected trees survive for several years before they are finally killed. To eliminate the inoculum potential, it was believed desirable to kill all trees of the white oak group which showed wilt symptoms.

White oaks showing wilt symptoms have been consistently destroyed throughout the program. The number of white oaks infected annually does not appear to be affected by the eradication program, but the number does appear to fluctuate up and down in alternate years.

Of the different herbicides tested, 2,4,5-T at a concentration of 16 pounds acid equivalents per 100 gallons of fuel oil gave the most satisfactory results for killing small oaks. Water soluble 2,4,5-T and CMU were not effective in giving complete kill. CMU was very slow in producing kill when used as a soil application around large trees. Chemical injury on other tree species was sometimes produced great distances from the zone of application. After being treated with CMU, trees often showed chemical injury for two to six years. The foliage symptoms caused by CMU were often confused with oak wilt when making aerial surveys. This made it necessary to spend additional time to carefully inspect each area from the ground.

Sodium arsenite gave the most satisfactory results in producing a rapid kill of infected and healthy trees. Although the amount of root-kill was not as great as would be desired, sodium arsenite did produce some root-kill, and the amount of kill obtained was possibly as much as could be obtained with any chemical applied in frills. Sodium arsenite practically eliminated the formation of mats. It is not believed essential to treat the trees in an early stage of wilt to prevent mat formation, although early treatment certainly would be desirable. Beetle infestation of infected trees treated with sodium arsenite also appeared to be reduced for at least one year after treatment. Translocation of arsenic in the vascular system is essential if any degree of insect-kill is to be expected. It was found necessary to treat trees in an early stage of wilt to obtain a sufficient amount of uptake of the chemical. If the wilt symptoms had become advanced, plugging of the vessels prevented translocation of arsenic to branches and sometimes

to the upper trunk portion. Bark on trees tended to slough about four to six months after treatment. Early sloughing of the bark is, of course, an advantage, since drying out of the wood should reduce or eliminate formation of fungus mats and insect activity.

Since Sinnissippi Forest has a very high population of deer and other wildlife, it was expected that some animal poisoning would occur with the use of sodium arsenite. However, during the five-year period in which the chemical was used, no such accidents were observed in the forest. The animal repellent incorporated in the sodium arsenite is largely responsible for reducing the chances of animal contact. It is believed that careful application of the chemical to prevent excessive runoff also reduced the poison hazard. It is not recommended that this chemical be used around livestock, although the danger of accidental poisoning can be eliminated by spraying a heavy application of creosote or applying roofing tar to all poisoned areas. Where only a few trees are to be treated in a small woodlot, the frills close to the ground can be covered with soil or sheet metal.

The two chemicals, Vapam and methyl bromide, gave encouraging results for killing oak roots in a limited application. The use of either chemical offers a new method of controlling spread of the oak wilt fungus, particularly on the property of homeowners. Methyl bromide is very toxic and consequently hazardous to use. Vapam, on the other hand, is quite safe if handled with care and may be easily applied.

SUGGESTIONS FOR PLANNING OAK WILT CONTROL IN FORESTS WHERE ANNUAL CUTS ARE NOT PROFITABLE

Each year approximately two weeks after the second oak wilt control work was completed, salvage operations were carried out. All merchantable timber killed by oak wilt or poisoned in establishing barriers was salvaged. Four men and the junior author were used in this operation. Studies conducted in 1959 indicate that the labor costs to produce and deliver 1,000 board feet of logs from the scattered oak wilt areas is 14 percent higher than the cost of normal logging in Sinnissippi Forest. Adding this same percentage to the supplies, overhead, and depreciation, the actual cost per 1,000 board feet of oak wilt logs delivered to the mill is $35.63. It has been determined from local mills that the value of such logs delivered to the mill is about $35 per 1,000 board feet. Since Sinnissippi Forest operates its own mill and can make a profit from the total operation, it is more desirable to take

this small loss in logging operations than it would be to let the trees stand for the regular 10-year rotation cut.

Small woodland holdings can be economically operated by logging once every five years. It is important that the trees removed do not exceed the five years' net growth. To be certain that the proper trees are being logged, the services of a forester should be obtained. In Illinois the name of the nearest forester can be obtained from the State Division of Forestry or the county farm adviser. In most states this service is available, as in Illinois, at no cost to the landowner.

Oak wilt areas must be treated at least twice each year. In small forested areas, the trees would not need to be cut until the five-year cut of the entire forest. If sodium arsenite is used as the poisoning agent, however, the treated trees dry out rapidly, and the bark sloughs off and some rot develops over a period of five years. Also, sodium arsenite should not be used in areas where livestock may come in contact with the chemical. Instead, 2,4,5-T may be used as a substitute where danger of contact may exist.

Using the five-year logging cycle, the cut will consist of the accumulated five-year net growth of the forest plus all poisoned and dead trees. In the average forest in Illinois, this should produce a volume of at least 1,000 board feet per acre, which is considered an economical cut by most sawmill operators.

SUMMARY

An experimental program to control the oak wilt disease was carried on in the Sinnissippi Forest in northern Illinois. Experimental control measures were tested for seven years to determine their effectiveness in preventing further spread of the disease. The study was designed to determine the amount of natural spread of the disease and the timber loss that might occur over a definite period of time.

To carry out an effective program, it was necessary to make either ground or aerial surveys two times a year. To prevent further spread of the disease, it was essential to treat infected trees as early as possible.

In the forested area, the number of trees infected annually through root grafts remained at a relatively low level during the last three years of the study. Where only currently infected trees and surrounding healthy trees were poisoned, the amount of root-graft transmission was reduced to less than 10 percent of what it was previous to treatment. In wilt areas where poison barriers were established around currently infected trees and around all infected trees killed in the previous three years, a substantial reduction in new infections by root-graft transmission was noted.

Overland spread of oak wilt in Sinnissippi Forest remained constant in one area and increased slightly in another area, although the number of trees infected through grafted roots decreased. Only seven trees were observed on which fungus mats were formed during the last three years of the study. This fact suggests that there are sources of inoculum other than fungus mats.

White oaks showing wilt symptoms have been consistently destroyed for the past five years. The number of white oaks infected annually does not appear to be affected by the eradication program, but it does appear to fluctuate up and down in alternate years.

Approximately 91 percent of currently infected trees of the red oak group were within 30 feet of trees previously killed by the oak wilt fungus. In untreated areas nearly one-third of the 279 infection centers studied were active after five years. Thirty-three percent of the single-tree infection centers did not repeat in six years of observation, and only 1 percent of all centers which contained five trees or less were active after five years. Periods between the appearance of new infection in individual oak wilt centers varied from one to six years. Therefore, if poison barriers are placed around only currently infected trees, it is necessary to re-treat some infection centers a number of times to completely stop further root-graft spread. Control was obtained in 71 percent of the areas where barriers were placed around only currently infected trees. Also, it was necessary to re-treat 24 percent of the infection centers a second time and 5 percent a third time.

Poison barriers were established on the earlier assumption that root-graft spread is dependent upon the size and distance between diseased trees. The maximum distance of root-graft spread when based on the size of trees was slightly less than the estimation made at the beginning of the study.

Sodium arsenite proved very effective in killing oak trees. Trees were killed more rapidly with sodium arsenite than with 2,4,5-T, Ammate, or CMU. Mat formation by the oak wilt fungus and insect activity were limited or prevented, and some root-kill occurred with the use of sodium arsenite.

In the seven-year program poison barriers were placed around 918 currently infected trees of the red oak group which were 4 inches in diameter and larger. The average diameter of the treated infected trees was 11.9 inches. In the entire program only 14 trees became infected beyond the barrier zone. With the establishment of poison barriers, an average of 2.9 healthy trees was poisoned for every infected tree.

In Sinnissippi Forest approximately 222,000 board feet of mer-

chantable timber were killed by oak wilt in the years 1945-1955. This is equivalent to an annual average loss of 8.0 percent of the total annual production of merchantable oak timber. This loss was reduced in five years to 3 percent of the annual production through the use of control practices.

Six chemicals, Vapam, methyl bromide, sodium pentachlorophenate, copper naphthenate, CMU, and Monsanto soil sterilant CP376, were tested on a small scale to determine if the roots could be killed in a limited zone by applying the chemicals to the soil. Both Vapam and methyl bromide killed all roots as large as 3 inches in diameter and to a depth of 3 feet. No chemical injury occurred on adjacent untreated trees. The remaining four chemicals were ineffective.

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